

CHALLENGES TO TAXONOMIC SYSTEMS FOR MENTAL ILLNESSES

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The field of psychiatric medicine is observed to lag far behind physical medicine. The notion of how mental illness ought to be defined is still debated. This confusion reflects serious problems in the system by which mental disorders are classified. The utility derived from the DSM-IV is, in some ways, undermined by the lack of scientific validity seen in the criteria employed therein. This paper suggests the use of a dimensional taxonomic model, onto which categorical criteria might be selectively superimposed. With such a system in place, a guide that expresses both utility for the clinician and validity for the researcher might be found.

Several years ago British psychologist Richard Bentall submitted an article to the *Journal of Medical Ethics*. The purported subject of this article was to classify happiness as a mental disorder. Although it is highly doubtful that Bentall's work was not recognized in the psychiatric community for the satire that it was, one must wonder how well this satire was appreciated. Bentall applied contemporary standards used for classifying psychological disorders to happiness. Although his argument was not without flaw, he made an interesting case for supporting the absurd in a simple attempt to call attention to the fact that our definition of psychological disorders is quite vague. There is considerable controversy today over whether or not our system of categorical taxonomy for the organization of psychological disorders is appropriate. Part of this trouble reasonably stems from the fact that it is difficult to specify exactly what we mean by psychological disorder. The goal of this paper is therefore tripartite: to examine the controversy surrounding the current definition of psychological disorder, to scrutinize the reliability and validity of our current taxonomic system, and to consider alternatives to it.

The Etiology Enigma: What Is Mental Illness?

Defining what we mean when we say that an individual has a "psychological disorder" is a much more difficult task than it might seem at first blush. The root of this controversy may, in fact, come from the fact that modern psychiatry stems from physical medicine. Physical medicine (i.e. allopathic medicine outside the realm of psychiatry) has progressed to a point where the etiology of physical disorders can be identified with relative certainty. For example, one might be suffering from tuberculosis if one has impaired breathing and presents with a productive cough (including blood in the sputum). Although the signs and symptoms (syndrome) the patient presents might be shared by lung cancer, physical medicine has some relatively straight-forward ways of assessing the different etiologies and deter-

mining illness. The fact that modern psychiatry is based on physical medicine predisposes it to seeking the same kinds of hard answers when looking to define mental illness. Psychological disorders, however, are not currently understood well enough such that a certain physiological marker can be identified as out of a "normal" range, thus indicating disease. When one examines a patient suffering from mental disease, at best one can observe a coherent syndrome, oftentimes without the benefit of thoroughly understanding its etiology.

With an understanding of the considerable ambiguity surrounding the etiology of mental diseases, definitions of mental "illness" must be found without the benefits of the physiological markers employed by physical medicine. Multiple theories have been proposed, some of which will be briefly discussed here to illustrate an inherent difficulty underlying the taxonomic process. However, the complete arguing of this particular point would merit an entire paper in itself. Ossario (1985) proposed that the inability to complete intended tasks and participate normally under social circumstances characterized mental illnesses. While definitions like Ossario's are intended to be broadly inclusive, they also fall prey to being too broad, a point well illustrated by Bentall's aforementioned satire. More formally, Wakefield (1997) criticized Ossario for being overly inclusive and that none of the defining elements are necessary or sufficient for the classification of mental disorder.

Wakefield proposed his own definition for a mental disorder in 1992, and while it seems to be an improvement on Ossario's, it is not without its own faults. Wakefield's proposed definition of mental illness involves "harmful dysfunction." "Harmful" refers to impaired activity as compared to the norm. "Dysfunction" refers to the absence or dysfunction of a normal biological mechanism that is related to mental function. These biological mechanisms are described in further detail by Wakefield; he asserts that these have been shaped by evolution. Although Wakefield's lan-

guage does highlight the underlying difficulties at hand (as noted by Spitzer, 1997), it is far from universally accepted. Lilienfield & Marino (1995) have argued that psychological traits aren't the results of evolution – they are instead the by-products. They posit that natural selection often leads to a good deal of variability among individuals (rather than phenotypic uniformity) and that many disorders are simply positive adaptations that are overly expressed in the patient. For example, anxiety is a positive adaptation in the sense that organisms with a healthy anxiety mechanism avoid potentially life-threatening situations. Anxiety disorder is simply an over-amplification of a positive trait.

Other problems with such a definition certainly exist – the “harmful” effect of a disorder requires the diagnostician to make an assessment that is quite subjective, and might well vary considerably from clinician to clinician. This decreased reliability does not necessarily imply that the validity of the diagnosis or the resulting treatment will be substantially better or worse, but it is certainly a danger. Perhaps more troubling is the “dysfunction” in question. Wakefield refers to the dysfunction of the underlying biological correlates of illness, and this would be fine if we knew what those biological processes often were. However, as noted, many mental illnesses are understood rather poorly, if at all, in regard to their etiologies. Wakefield's definition of mental illness shows the penchant for the objective that harkens back to psychiatry's roots in physical medicine.

Given the problems inherent in accurately defining something as contentious as psychological disorders, the DSM-IV presents a set of guidelines to be considered in the determination of whether a mental illness is truly present. These guidelines include statistical abnormality, the violation of social norms, personal distress, and disability/dysfunction. These four points, each of which is necessary but not sufficient for diagnosis of illness, are meant to be coupled with the observation that they are unexpected in given circumstances – the idea being to provide the inclusiveness that Ossario sought without yielding inappropriate diagnoses. The DSM-IV guidelines also avoid basing a definition on unknown etiologies, as Wakefield's model does. However, the system is not perfect in its definition of illness, and an understanding of this fundamental instability is requisite to the exploration of the more complex topic of compiling the taxonomy of mental illnesses.

Partitioning the Continuum: Is Syndromal Taxonomy Valid?

Like the definition of mental illness itself, the taxonomy of such disorders cannot simply follow the format

of physical medicine. Again, a large degree of uncertainty in regard to the etiology of mental illnesses prevents one from grouping disorders based on their underlying causes. Instead, we are presented with clinical signs and symptoms. The same (unknown) etiology may well lead to myriad manifestations. Conversely, the same clinical signs and symptoms may originate from very different etiologies. Without the luxury of a comprehensive understanding of etiology, the only reliable way of grouping disorders is on the grounds of symptomatology.

The current taxonomic system is an organization of discrete syndromes, and it has certainly increased the reliability with which mental illnesses are diagnosed. This should be seen as a crucial first step, for uniformly effective treatments or correlative factors for a disease can only follow reliable diagnosis. To demonstrate the increased reliability seen in modern classification, Hasin et al. (1996) performed a test/re-test of patients with dual diagnosis or substance abuse problems. Their test/re-test comparison revealed excellent reliability in the diagnosis of these patients for whom diagnostic reliability had been a problem before the advent of DSM-IV. Similarly, Fennig et al. (1994) used the DSM-III to assess 6-month stability in patients diagnosed with psychosis and schizophrenia, finding 87-89% of patients staying in the same broad category, with only 62-68% staying in the same sub-category. Additionally, Sartorius et al (1995) found that inter-rater reliability was high for the diagnosis of most categories, but at the sub-type level only half of the categories showed excellent agreement. From the aforementioned studies, one can gather that the syndromal taxonomy of mental illness has at least increased the reliability of diagnosis in general terms, although the diagnosis of sub-types may be more difficult as less research has been done in these more specific areas.

Less promising results regarding reliable diagnosis have been found with some Axis II (personality) disorders. A good illustration of this is the Nelson & Rice (1997) study in which they tested 1-year stability in patients originally diagnosed with obsessive-compulsive disorder (OCD) – only 19% of patients were re-diagnosed. This seems a far cry from the relatively heartening support of increased reliability seen above. But might this fluctuation be characteristic of the type of disorder seen here? One can only surmise that different etiologies exist for different types of mental illnesses. While schizophrenia, which research suggests is at least partially biological in origin, has a relatively reliable diagnosis, it is reasonable to give credence to the idea that personality disorders may only become evident when a particular stressor is applied to an individual who, through

personality disposition, is prone to crossing that ambiguous threshold into “disorder.” Given a change in circumstances over year-long course of this study, what was a personality disorder might well fade into “normality” while the predisposition remains. The disorder could certainly recur at some point in the future, should the appropriate stressor(s) be applied. Support for this idea will be later mentioned in this paper under the topic of dimensions of personality and disease.

The validity of disorders under our current taxonomic scheme seems, unfortunately, to be as full of caveats as is the reliability. It is important here to distinguish between validity and utility. What the clinician means by “validity” may be quite different from what the researcher means in using the same term. In a discussion of scientific validity, a disease-entity is only (according to Kendall and Jablensky, 2003) valid if one of the following two categories is met:

- 1) If the defining characteristic of the category is a syndrome (group of symptoms), this syndrome must be demonstrated to be an entity, separated from neighboring syndromes and normality by a zone of rarity.

- 2) If the defining characteristics are more fundamental (defined by a physiological, anatomical, histological, chromosomal, or molecular abnormality), clear qualitative differences must exist between these defining characteristics and those of other conditions with a similar syndrome.

Two points must be made in regard to the above guidelines. The aforementioned “zone of rarity” refers to the lack of continuous variation between two similar syndromes. If one syndrome contains a certain set of symptoms, then a similar syndrome is said to be independent if and only if the prevalence of a middle ground, a syndrome composed of some symptoms shared with the first and some symptoms shared with the second, is very rare. This is often not found across the spectrum of mental illnesses (Kendall & Jablensky, 2003). The defining characteristics referred to in the second point are reflective of Andreasen’s additions (molecular genetics & molecular biology, neurochemistry, neuroanatomy, neurophysiology, and cognitive neuroscience) to Robins and Guze’s original validating criteria (clinical description, laboratory studies, delimitation from other disorders, follow-up studies, and family studies). Scientific progress is being made in elucidating the etiologies of numerous mental illnesses, albeit slowly. Faraone et al (1995) reviewed 30 studies of putative genetic indicators of schizophrenia, and found that only 6 turned up results that improved the no-

tion of a genetic etiology. The aforementioned results call attention to the amount of difficulty encountered in trying to validate conceptions of mental illnesses in the scientific sense.

Some (or most) of the DSM-IV categories might not be valid in the scientific sense that they aren’t discrete disease entities either separated from one another by zones of rarity or distinct etiologies. However, they do seem to be invaluable for clinicians; that is, they have great utility. The DSM-IV defines utility (cited in Spitzer, 2001) by the helpfulness of a category and the information provided about the disease in terms of diagnostic power, prognosis, treatment plans, and the like. We can therefore see where a problem might arise with the DSM-IV, commonly used by both researchers and practicing clinicians- two groups that often have very different needs in terms of “validity.” For the diagnostician, the current DSM-IV is valid in a sense of utility, for many researchers, problems arise because the DSM assigns arbitrary cutoffs to what might better be described as a continuum of dysfunction. As this manual guides research to a degree, more pertinent research should be done on deciphering whether there are in fact some disorders that do have zones of rarity and distinct etiologies or whether they are, by and large, characterized by a continuum. Researching a disease that is accurately characterized as a continuum of dysfunction as if it were simply syndromal is inefficient. Herein one sees the benefit of having different versions of a reference for clinicians (where validity in a utility sense is important) and researchers (where validity in the scientific sense is paramount) – the ICD-10 is a good example of a reference that avoids some of the problems seen with the DSM-IV .

Dimension and Dysfunction: Alternatives to Syndromal Taxonomy

It is possible that, if the vast majority of disorders are best represented by dimensions of disease, illness should be classified dimensionally rather than by syndrome. Doing such would at least relieve the need for separate clinician/research manuals. But is this appropriate? Support for a dimensional system of classification, at least in terms of personality disorders, comes from work done by Hans Eysenck. Eysenck put forth a model of personality that consisted of three factors: introversion-extraversion, neuroticism, and psychoticism. Each individual falls somewhere along a continuum in relation to each of these three traits. Eysenck’s work was based on Ivan Pavlov’s, who noted differences in the excitability of the dogs he was training. Eysenck used this excitability difference in dogs to underpin his theory

that each individual has a different nervous system, and as a result some will possess nervous systems that are more easily excited while some possess nervous systems that are more easily inhibited. Eysenck posited that introverts have nervous systems that are more easily excited (via the reticular formation), while extraverts' nervous systems are more easily inhibited. This theory clearly lends itself to a dimensional range of excitability based on the relative strengths of the inhibitory/excitatory mechanisms in the brain. The importance of the introversion-extraversion range is underscored when taken in conjunction with the fact that Eysenck surmised it to be explanatory of variation within the fields of neuroticism and psychoticism (Claridge, *Origins of Mental Illness*).

Eysenck's dimensional model of personality seems logical enough, and the biological underpinnings of his theory have been supported by evidence. But for a theory of this nature to have any influence on the taxonomic organization of mental illnesses, clinical observations are required in addition to organized laboratory experiments. These clinical observations have come in the form of high comorbidity rates among personality disorders. Personality disorders have an astoundingly high comorbidity rate that could easily be explained by Eysenck's three-dimensional model of personality. With each individual expressing a particular (x,y,z) coordinate in three dimensions of personality, it is rather straight-forward to surmise that if any one of these variables is sufficiently deviant from the average, multiple personality disorders (different manifestations of the same wayward variables) could occur. The shades of grey that result have been indicated clearly by clinical research. Minor differences in the definition of major depression, to provide one clinical example, greatly influenced its prevalence in study populations (Kendler, 1988; Regier, 1998).

With the advent of more complex scientific methods of exploring the neural underpinnings of mental illnesses, it seems clearer that not all of them are the result of aberrant personality traits – some do indeed have valid biological roots. This is not to say, however, that the cause of a given mental illness is either personality based or biologically based. It seems far more rational to examine mental illnesses in the context that each person has a certain personality, which may predispose one to a certain illness. Taken in hand with a possible biological predisposition and the presence of environmental stressors (should the biological and personality predispositions be mild enough to not cause dysfunction themselves), disease may manifest. As an example, Nathan (1993) found that alcohol abuse and dependence manifested differently based on differing personality traits. Given the

idea that the causation of mental illnesses might be a more complex web than thought, it seems foolish to assign either a dimensional or a categorical designation exclusively to all illnesses.

Some illnesses, like schizophrenia, may in fact require both. Schizophrenia is categorically differentiated from affective disorders, although Kendall (1975, in Cooper & Cooper *Adult Abnormal Psychology*) has found that phenothiazines are the most effective treatment for both and that the same range and probability of outcomes exists for both disorders. This is highly indicative of the fact that schizophrenia and some affective disorders may just be different areas on one dimension. But schizophrenia should not necessarily be seen to be completely dimensional for we still do have some very useful categories in determining its sub-types. Different sub-types have different, all-or-none categories (e.g. one hears voices or one does not). The fact that the affective side may be best viewed as a dimension, on top of which one can super-impose further sub-categories is only further supported by the fact that those categories may differ in themselves (according to things like the frequency with which one hears voices, the severity of the statements they make, etc.). The best system may be one in which there are numerous categories and dimensions for more complex mental illnesses while others may be simply personality-based (Clark, 1998, suggests a complex hierarchy of categorical and dimensional classification).

Conclusions

There are numerous implications of the taxonomic system on treatment. Rachman & Philips have argued that it is detrimental for patients to be labeled with a mental illness because of the associated societal stigma (Rachman & Philips, 1978; Rachman & Wilson, 1980, in Cooper & Cooper *Adult Abnormal Psychology*). No doubt this stigma comes from a poor understanding of what causes mental illnesses, and should the etiologies of psychological disorders come to be more fully understood, these stigma may recede considerably. Until that time and in spite of stigma, so long as identification is conducive to treatment, clinicians are ethically bound to denote full record of a patient's illness (kept confidential, of course). Yet other practical problems regarding treatment exist as well. Rosenham's 1973 study, where he and a number of colleagues voluntarily faked mental illness so as to be committed, reveals a rather unsavory state of the facilities for the treatment of the mentally ill. The isolation and prison-like environment to which the mentally ill are subjected is no doubt pathological in and of itself. Might this denote the need to return to facilities such as the

moral hospitals of the nineteenth century, in which patients were treated as normal people with temporarily illnesses? Housing patients in a more comfortable setting where they can be intellectually stimulated may be highly helpful in their recovery. Even the medieval practice of circulating the mentally ill among relatives and members of their close communities had its merits – we can certainly assume that today’s institutions are stressors in and of themselves, and given the aforementioned personality predispositions, being in a comfortable environment might well help quell forceful manifestations of wayward personality traits.

To conclude, we find ourselves at a nexus in the field of psychiatry; a place where the possibility, rooted in empirical evidence, exists for overhauling our current syndromal taxonomic system of mental illnesses to include dimensions where appropriate. This effort has long been confounded by questions of theory (what is a “mental illness” after all?) as well as by questions of laboratory science. Nevertheless, demonstrable advances have been made in increasing the reliability of diagnoses, without which further advances would certainly be hampered. The bearing of etiology on true validity cannot be underestimated, and is not fully appreciated by our current syndromal taxonomic system. For example, McNally (1991) has shown that single incident stressors lead PTSD patients to relive their experiences while prolonged stressors lead PTSD patients to display dissociative symptoms. Conversely, we currently denote different diagnostic categories (GAD and major depression) for illnesses that have extremely similar, if not indistinguishable, neural correlates (Kendler, 1996).

The optimal taxonomy is clearly one, like physical medicine, that is based on classification by etiology. The most effective and efficient treatments only come from being able to remedy the underlying cause of disease, mental or physical, and so long as we are unable to group disorders based on etiology, these treatments will not be realized. We are currently unable to do this because of vast gaps in our understanding of mental illnesses, and research needs to be targeted to parse personality causes from biological ones, as well as in the further refinement of functional models for each group. We can conclude that alternatives to our current system might be preferable; in the temporary sense an integration of dimensions and categories would be beneficial, and in the long-term, a thorough development in our understanding of etiology is imperative.

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